THE SYMPTOMATOLOGY OF LESIONS OF THE LENTICULAR ZONE WITH SOME DISCUSSION OF THE PATHOLOGY OF APHASIA.

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THE ROLE PLAYED IN THE PRODUCTION OF APHASIA BY LESIONS OF DIFFERENT PARTS OF THE FIRST TEMPORAL CONVOLUTION.

In the discussion of the effects of lesions of the lenticular zone and the zone of Wernicke* in the production of aphasia, the importance of each portion of these zones must be carefully considered. It is only in this way that we can arrive at reliable conclusions on the mooted questions of the localization of the representation of the different elements of cerebral speech. The first and second temporal convolutions necessarily hold a prominent place in this discussion. It has been usually held that the higher auditory area, the so-called center for word hearing, is situated in the posterior portion of the left first temporal convolution, or of the first and second temporal convolutions, the focus of representation being about opposite the posterior upward turning of the horizontal branch of the Sylvian fissure. As a rule more stress has been laid upon lesions of the cortex than of the subcortex in the causation of word deafness, although of course the pure word deafness of Lichtheim and Dejerine has been attrib-

In the two cases, the histories of which have just been given, utable to injury of subcortical tracts proceeding to the cortical center from the basal centers and the auditory periphery. word deafness was undoubtedly present and somewhat persistent, although not permanent. In both of these cases the zone of Wernicke, as described by Marie, was not involved in the destructive lesion, this being confined to the white matter of the anterior portion of the first temporal convolution.

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*It might be well to give here Marie's definition of this zone. According to him it consists of the supramarginal and angular gyri and the feet of the first two temporal convolutions. Its limitations cannot be accurately determined, and it is impossible to say whether the symptoms accompanying a lesion of this zone are caused by the alteration of the cortex or the subjacent white matter; Marie inclines toward the importance of the white matter.

It is not necessary to call into service the theory of diaschisis of von Monakow to explain the word deafness in these cases. When word deafness is a symptom, the subcortex of the anterior portion of the first temporal convolution being diseased, while the cortex and subcortex of the posterior halves of the first and second temporal convolutions escape, the aphasic phenomenon finds its explanation in destruction of the paths of transmission to and through the external and extreme capsules to the insula and the center of Broca. One of the writers, Dr. Mills,[20] has reported a case, now well known in the literature of the subject, which shows that word deafness may result from a lesion limited to the posterior portion of the first or of the first and second temporal convolutions, chiefly to their cortex, and reference will next be made to two cases previously recorded by Dr. Spiller.[21]

Case 3. *Fracture of the Skull in the Temporal Region—Contusion and Disintegration of the Inframarginal and Supramarginal Regions—Hemiplegia, Hemianesthesia and Aphasia Nearly Total at First—The Disappearance of Paralysis and Improvement in Motor Aphasia, Marked Word Deafness and Paraphasia Persistent—Operation with Improvement—Relapse—Auditory and Visual Hallucinations—Persecutory Delusions—Necropsy Showing Destructive Lesion Chiefly Involving the Zone of Wernicke (the Supramarginal and the Posterior Portions of the First and Second Temporal Convolutions*).

The patient, a man twenty-nine years old, had suffered from a fracture of the skull which left him at first completely hemiplegic and hemianesthetic on the right side and almost totally aphasic. He recovered largely from the paralysis and to some extent from the speech disturbance, but word deafness and paraphasia were pronounced. He was probably word blind.

An operation revealed a stellate fracture with contusion and disintegration of brain substance, probably of the first and second temporal convolutions. Word deafness persisted, and later the patient became insane with auditory and visual hallucinations and delusions of persecution. The patient died in the insane department of the hospital and necropsy showed an area of sclerosis involving the posterior part of the first and second left temporal convolutions, and also part of the parietal lobe. It extended to the posterior part of the insula and to the optic radiations.

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[20] Mills, Chas. K. "Lesions of the Superior Temporal Convolutions Accurately Locating the Auditory Center." University Medical Magazine, Vol. 4, November, 1891.

[21] Spiller, William G. "Lesions of the Left First Temporal Convolution in Relation to Sensory Aphasia." Review of Neurology and Psychiatry, May, 1906.

The necropsy showed the reverse picture of the lesions in cases 1 and 2 so far as the temporal lobe was concerned, the sub-cortex of the anterior halves of the first temporal convolutions being destroyed in these cases. Word deafness was pronounced but not complete. It would seem, therefore, from these three cases that either a destruction of fibers passing from the posterior part of the left first temporal convolution by way of the external capsule, or of these fibers plus a lesion of the cortex of the posterior part of the left first temporal convolution may cause word deafness. In the first two of these cases the word deafness was less persistent. It is to be noted that the sclerosis in case 3 involved the upper part of the middle portion of the left second temporal convolution.

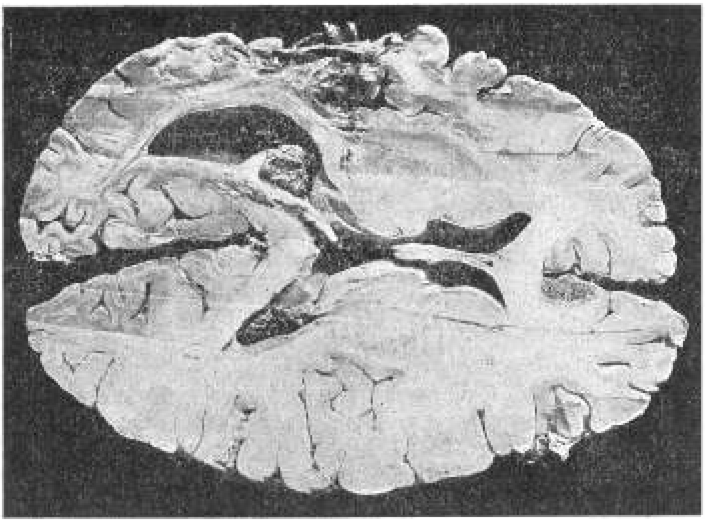

Fig. 4. Complete destruction of the first temporal convolution, with marked sensory aphasia. The lesion extends to the lenticular zone. (Case 3.)

Case 4. *Apoplectic Attack twenty-three years before death causing Hemiparesis and Disorder of Speech—A Second Apo-*

plectic Seizure fifteen years later—Slight Paraphasia and Paragraphia (Occasional Omission or Misuse of Words in Talking or Writing)—Word Deafness not present for several years before his death—No Facts as to Word Hearing at the Time and immediately after his aploplectic attack—No Word Blindness or Letter Blindness—No Mental Impairment except some Defect of Memory—Was Ataxic and had other symptoms of Tabes—The Microscopic Findings of Tabes—Destruction of the Entire Left First Temporal Convolution except in the Angular Region—Destruction of the Insula except at its anterior pole—No Involvement of the Lenticula.

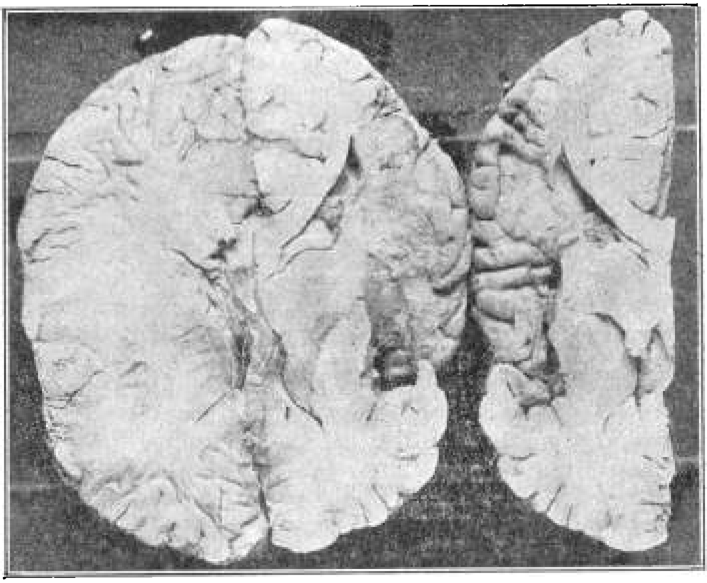

Fig. 5. Complete and long-standing destruction of the left first temporal convolution without marked persistent sensory aphasia. The destruction extends to Marie's lenticular zone. (Case 4.)

A man, about sixty-one years of age at the time of his death, twenty-three years before this time had an apoplectic attack. He was left with some disorders of speech and impairment of power in the right half of the body. About eight years before his death he had a second seizure. He became partly aphasic, could not write without making mistakes, walked with difficulty, and at times had difficulty in swallowing and in urination. He also had spells of hiccough and vomiting. He had cramps or twitchings in the right leg.

The patient, at least in a late period in the history of his case, had no difficulty in understanding words. He was sometimes at a loss for a word, although he could talk with comparative ease. He was liable to misuse words in writing, sometimes misspelling, but these defects were but little marked. He was sound mentally, except that his memory had failed somewhat. He was right-handed. He read constantly, evidently not being word or letter blind. He was ataxic and had other symptoms of tabes.

The left first temporal convolution was destroyed in its entire length except at the angular and supramarginal gyri. The sclerosis extended to the posterior horn of the lateral ventricle. It destroyed the cortex of the island of Reil and the white matter immediately subjacent except in the extreme anterior end of the island. It did not involve the lenticular nucleus. The lesion was very similar to that in the brain of the patient in case 3, except that it extended a little more inwards and considerably further forward, but left the supramarginal and left second temporal convolution intact. It did not cause persistent word deafness, at least the patient was not word deaf at the time his case was studied. About his condition as to word hearing at the time of his attack and in the earlier years after it, no record was made.

In the original report of this case, one of us, Dr. Spiller, has suggested that the only explanations for the preservation of word hearing in this case are that the center for this function was largely in the posterior part of the left second temporal convolution, or that the right first temporal convolution had been unusually well developed during the patient's entire life, and was capable of assuming the function of the destroyed left first temporal convolution. It seems improbable that the small portion of the angular and supramarginal gyri preserved could explain the retention of word hearing.

The preservation of mentality in this case, as in case 2, is of interest in connection with Marie's views as to intellectual deficit caused by lesion of Wernicke's zone. The history of the case emphasizes the fact, to which attention has been directed by one of us[22] in a recent paper, that the degree of intellectual deficit in cases of lesion of this zone is largely conditioned by the intelligence and general capacity of the patient before the attack causing the aphasia.

THE INSULA AS A DISTINCT AND IMPORTANT FUNCTIONAL AREA.

One of the results of the acceptance of the views of Marie is to underrate the importance of the insula as a functional area or as a portion of a functional area concerned with speech. The effort should in the first place be made to distinguish between the functions of the insula and those of the lenticula.

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[22] Mills, Chas. K. THE JOURN. OF NERV. AND MENT. DIS., Vol. 34, July, 1907.

Some of the contributors to our knowledge of aphasia, and especially some of the earlier writers of the period beginning with Broca's discovery in 1861, have gone so far as to regard the insula as the sole cortical motor center for speech, a view which must fix our attention in connection with some of the findings given in this paper, although we are inclined to the opinion that it is only a part, but it may be a large part, of this cortical motor center. The numerous cases of lesion of Broca's convolution with motor aphasia, even if the lesion in many of these cases was not confined to this convolution, cannot be set aside. In one of our own cases (case 5) the lesion causing the motor aphasia was nearly equally distributed between the insula and Broca's convolution, Wernicke's zone, the lenticula and the capsules not being involved.

For the literature of the insula in its relations to speech we must refer our readers to such well-known works as those of Bateman, Wyllie, Dejerine, and others.

Dr. E. A. Spitzka[23] has contributed something of interest in this connection from the standpoint of the anatomist and morphologist. He has directed attention to the redundancy of the preinsula in the brains of highly educated men,—men with great capacity for language both as a means of verbal expression and of reasoning.

The island of Reil is embryologically very different from the striatum. The latter rises from the base of the brain and is separated by a fissure, as Edinger says,[24] from the mantle or pallium, whose wall becomes thickened relatively late. The striatum develops in all vertebrates, but it is only in the higher that the pallium has a development of importance. The pallium later contains the cortex, on which depends all mental processes. In a figure of a frontal section through the brain of a human embryo at the age of two and one-half months, given by Edinger, the striatum rises free from the base of the brain, distinctly separated by a fissure from the external wall of the lateral ventricle. At a later embryological period (about the fourth month) it is divided into the caudate and lenticular nuclei by the fibers of the internal capsule growing through it. The caudate nucleus projects free into the lateral ventricle, as does the lenticular nucleus also; but in later embryonal life the small fissure between the latter and the wall of the cerebral hemisphere becomes so narrowed as to be invisible; but even in adults the wall of the hemisphere can be separated from the outer part of the lenticular nucleus without tearing of fibers. This region of the early fissure is sometimes of importance in the fully developed brain. Hemorrhage occurs here very readily, and the blood, if not too great in quantity, fills the space between the wall of the cerebral hemisphere and the outer division of the lenticular nucleus. Edinger believes that the striatum has some of its fibers in the anterior limb of the internal capsule.

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[23] Spitzka, E. A. Amer. Anthropologist, N. S., Vol. 5, October-December, 1903.

[24] Edinger, L. "Vorlesungen über den Bau der nervösen Centralorgane."

These remarks of Edinger explain why the external capsule is so often the area occupied by a hemorrhage or a cyst. In one of our cases (case 6) a cyst was found in each external capsule which was a *locus minoris resistentiæ.*

In another case studied by us,—not included formally in the cases given in this paper,—in which the lenticula was in an early stage of red softening, no separation of the lenticula from the island of Reil was seen when the brain was first opened, but during the process of hardening the putamen contracted from the white matter of the island, leaving a fissure in the external capsule.

The blood supply of the lenticula and of the insula and its subcortex seems to be distinct. We have preparations from a case (case 7) in which occlusion of the middle cerebral artery occurred after the supply to the basal ganglia had been given off. The point of thrombus formation is clearly demonstrated. The softening implicates the island of Reil and subjacent white matter, but leaves the lenticula intact. The patient could not speak during three weeks, but seemed to understand.

From these facts we may conclude that the function of the island and its white matter is distinct from that of the lenticula, and cases in which both structures are implicated may be wrongly interpreted.

Case 5. *Apoplectic Seizure causing Aphasia without Paralysis of Face or Limbs—Aphasia of Motor Type—A Few Recurring Utterances—Marked Paralexia—Some Retention of*

Writing—No Impairment of Articulation or Vocalization—An Old Cyst Involving only the Cortex and Subcortex of the Caudal Portion of the Left Third Frontal Convolution and the Insula.

Althought the following case has been used by one of us, Dr. Mills,[25] in a previous article, and has also been referred to by Gordinier,[26] it has so much value on the negative side of the discussion of the functions of the lenticula, as this ganglion was not involved in the lesion, and also so much on the positive side of the consideration of the functions of the insula and Broca's convolution, both of which were destructively implicated, that we have thought it important to reproduce it in connection with our present series of cases. There is scarcely to be found in the literature of aphasia a more instructive case. Neither Wernicke's zone, the lenticula, nor the internal capsule was involved, and yet the patient showed motor aphasia of typical form with the retention of some power of writing and with the retention (or reacquirement, as the case was not seen at an early period after the apoplectic attack) of the power of reading silently.

The third frontal gyre and the insula were neatly involved in the same circumscribed lesion, the second frontal convolution, the internal capsule, the basal ganglia, and all other portions of the brain not being the seat of any old lesion.

The following citation and summary are taken from the paper in the *American Journal of the Medical Sciences*:

"This patient, after an apoplectic seizure, developed aphasia, apparently without preceding paralysis or any other symptoms of focal lesion like visual blindness and hemianopsia. When examined about nine years after the onset of the aphasia, he had almost complete inability to name persons and objects which he was able to recognize through all his special senses. He had also a marked form of paralexia. When he attempted to read, although he understood what he was reading, he repeated an absurd formula of a few phrases. He had limited spontaneous speech, even using short sentences without concrete nouns correctly. He could write many single words correctly, sometimes misspelling, however. He held his pen or pencil correctly and wrote with ease and firmness."

Many examinations of this patient were made. The case was clearly one of aphasia with marked verbal amnesia and general disorganization of language on its dynamic side. Grammatical expressions and combinations were lost. The patient could speak only in single words, or in short phrases, or very short sentences. What he said, however, he articulated and enunciated clearly.

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[25] Mills, Chas. K. Amer. Journ. of Medical Sciences, September, 1904.
[26] Gordinier, C. H. Idem, 1903.

He had no paresis of the organs of articulation, enunciation or vocalization.

The only lesion to be seen in the lateral aspect of the left hemicerebrum was confined to the hinder part of the third frontal convolution. The lesion was probably part of an old hemorrhagic cyst. When the brain was sectioned at the level of the insula the insula was found to be largely destroyed by a lesion which was continuous with that in the caudal part of the left subfrontal gyre. Other parts were not involved, as shown by this section and also a subsequent one made a little lower than the first.

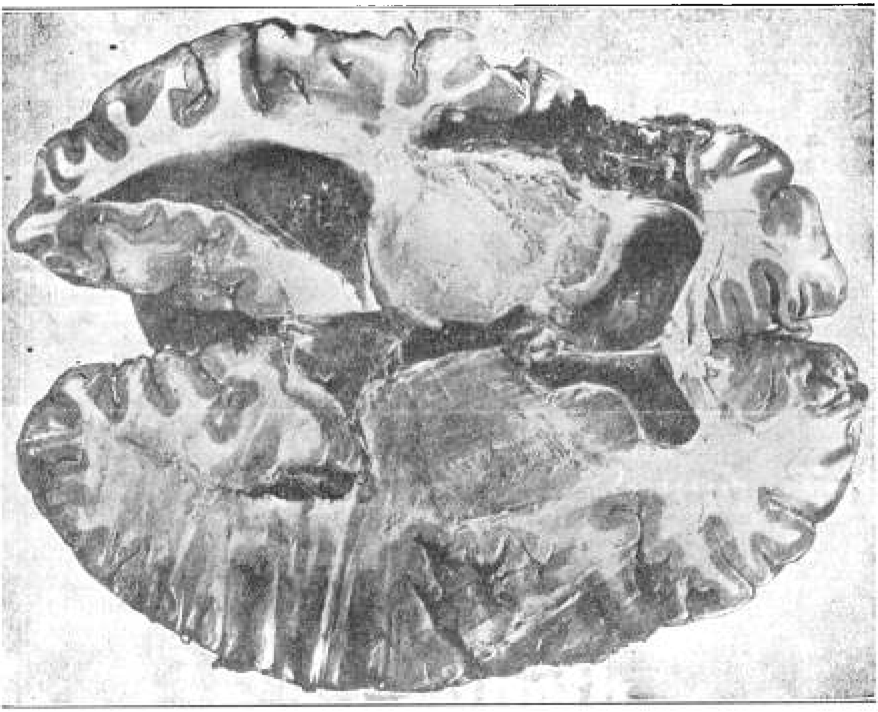

Fig. 6. Case of motor aphasia without hemiplegia. The primary lesion involves the left third frontal convolution and the insula. (Case 5.)

It is worth while to refer in this connection to one of the most recent and valuable contributions on the pathology of aphasia —a paper which forms a part of the general discussion which has arisen since the promulgation of the views of Marie. In this paper, in addition to many important considerations regarding focal lesions and the pathology of aphasia, Dejerine[27] gives the details of two previously unrecorded cases studied both macroscopically and in microscopical serial sections. In these cases the primitive lesions causing the aphasia were confined to the cortex and subcortex of Broca's convolution and to the second frontal convolution with a slight involvement of the insula and extreme capsule in one case. The zone of Wernicke, the lenticula and the capsules, except as mentioned concerning the extreme capsule, were not implicated if we exclude processes of secondary degeneration.*

The first of the two cases reported by Dejerine in this paper was one of motor aphasia, at first very marked, the patient retaining only "yes" and "no" and interjections. Any evidences of sensorial aphasia were very temporary. No paralysis, orolingual, facial or of the limbs, was present. The patient was motor aphasic without being dysarthric. Writing could not be tested, as the patient had lost his right arm by amputation and had not learned to write with his left hand. As time progressed, and before his death two years after the apoplectic attack, he had regained largely the power of speech.

The necropsy showed a lesion similar in some respects in its limitations to case 5 of our series, the insula, however, not being nearly so much involved. In Dejerine's first case the lesion destructively involved about two-thirds of Broca's convolution and to a very moderate degree the anterior portion of the insula and the related segment of the extreme capsule. The caudate and lenticular nuclei were not attacked by the primary lesion, as were not the Rolandic operculum and the external and internal capsules. Serial microscopic sections showed some involvement of the second frontal and of the corona radiata, with a considerable portion of the frontal lobe anterior to the precentral convolution. Degeneration was distinctly marked in the knee of the callosum. The temporal lobe was nowhere diseased. Softening was observed in the first and second frontal convolutions of the right hemisphere. The degeneration involved the anterior segment of the internal capsule, the fibers of passage through the striatum, and the occipito-frontal bundle. Degeneration of the anterior fibers of the extreme and external capsules was also present.

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[27] Dejerine. J. L'Aphasie Motrice et sa Localisation Corticale. L'Encéphale, No. 5, May, 1907.

*We understand by Broca's area, which has been described differently by writers on aphasia, that corticosubcortical area which surrounds the ascending branch of the Sylvian fissure, including its bifurcations. This makes Broca's convolution include what Dejerine calls the orbital portion, the cap and the foot of the convolution. The foot of this convolution is the part situated between the precentral convolution and the horizontal and ascending branch of the Sylvian fissure.

In the second case of Dejerine, the patient, a woman seventy years old, had a left hemiplegia and some years later an attack causing motor aphasia. This motor aphasia as described was typical and persisted. The patient retained only a few expressions, such as "yes," "no," and "good day." She was not word deaf, word blind or letter blind, or if word blind at all only to a limited extent. She understood all that was said to her, even when she was spoken to rapidly. She could read what was placed before her, but had difficulty if the sentences were long or complex. She could not write, except her name, although she held the pen correctly. She could, however, copy and transfer print into script.

We shall omit the lesions found in the right hemisphere in this case, except to say that they were such as to account for her left hemiplegia and perhaps some other phenomena. In the left hemisphere a yellow plaque was found, so situated as to destructively involve the cap of Broca's convolution, and also in part the second frontal convolution. Serial microscopical sections showed that this primitive lesion did not involve the insula, the external or internal capsules or the basal ganglia. The white matter of the third and second frontal convolutions was considerably diseased. Degenerations were present much as in the first case, involving, for example, the anterior segment of the internal capsule, the radiate fibers to the thalamus and the knee of the callosum. The zone of Wernicke and all other parts of the left hemisphere were intact.

This case clearly shows that Broca's aphasia (motor aphasia of persistent form) may be due to a lesion involving the third and second frontal convolutions and the subjacent white matter in the primitive lesion, all other parts escaping.

The first of the two cases is of similar import, although the insula and extreme capsule were to a slight extent included in the lesion, and the patient recovered largely from his motor aphasia. This recovery may have been due to the restoration of the parts of Broca's convolution and the insula not included in the lesion, or as Dejerine suggests, the fact that the patient had lost his right upper extremity some twelve years before his death may have had something to do with the unusual facility with which the convolutional areas of the right hemisphere, homologous to those of Broca in the left, assumed the representation of motor speech.

Case 6. *Right Hemiplegia with Exaggerated Reflexes on the Paralyzed Side—Attacks of Vertigo—Linear Cyst in the External Capsule of Each Hemisphere—Area of Sclerosis in the Middle of the Foot of the Left Cerebral Peduncle—Right Pyramidal Tract in Cervical Region Degenerated.*

The notes of the following case were meager.

E. D., aged fifty-seven years, white, German, was admitted to the Nervous Wards of the Philadelphia General Hospital November 14, 1902. He had been a strong man and had worked as a sailor.

When admitted to the hospital he stated that he was paralyzed on the right side six years ago. He said that he had had attacks of vertigo every day for the last six months, in some of which he fell to the floor. The eyes reacted normally to light and in accommodation. On the right (paralyzed) side knee-jerk was excessive, slight on the well side. No ankle clonus could be obtained, but a slight Babinski response was present on each side.

The next notes in this case were not made until September 11, 1903, when the records state that the patient was in a stuporous condition, unable to answer any questions and he did not seem to understand anything that was said to him. He had great difficulty in swallowing.

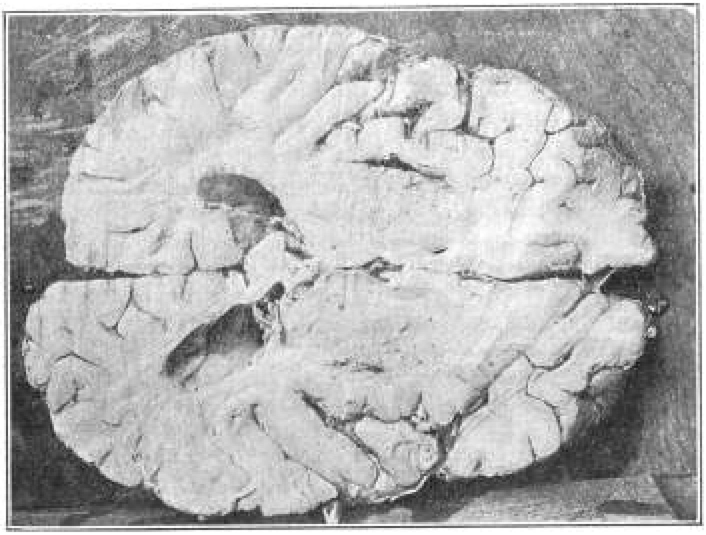

Fig. 7. A symmetrical cyst is present in each external capsule in the region of least resistance. (Case 6.)

On September 15, 1903, the notes stated: He became weaker this evening and was no longer able to be out of bed. Incontinence of urine and feces was present. He died September 20th. The fresh spinal cord seemed to show sclerosis of both right and left pyramidal tracts. The brain showed some areas of softening of the right caudatum.

A horizontal section through the cerebral hemispheres nearly at the upper border of the lateral ventricles showed a small hemorrhagic scar in the right hemisphere probably cutting some of the fibeers that form the knee of the right internal capsule. This may have developed shortly before death. A linear cyst was found in the external capsule of each hemisphere in a section made through the upper part of the thalamus and lenticula. The cyst on the right side to the naked eye measured 2.5 cm. in length and 0.5 cm. in its widest portion (1 in. in length by 1-5 in. in width). It did not extend into the cortex of the island of Reil and encroached very slightly on the lenticula. Its anterior end was 0.5 cm. (1-5 in.) behind the anterior end of the lenticula. Its posterior end reached very nearly to the posterior end of the lenticula. The cyst on the left side was smaller than that on the right side. It was in exactly a corresponding position except that its anterior end was 1.5 cm. (3-5 in.) behind the anterior end of the lenticula. Its posterior end extended to the posterior end of the lenticula. It also encroached very little on the lenticula. Neither cyst extended into the capsule, and neither extended downwards as far as the level in which the cerebral peduncles appeared distinctly formed, nor did they change their relative positions. No cause was detected for the right hemiplegia.

Sections of the cerebral peduncles and of the cervical region of the spinal cord made by Dr. T. H. Weisenburg showed a small area of sclerosis in the middle of the foot of the left cerebral peduncle. The right crossed pyramidal tract of the cervical region of the cord was degenerated. The lesion producing this degeneration has not been found, unless it was the cyst in the external capsule, which was doubtful.

The history in this case is very meager, but we have retained it in this series because the notes record that the patient stated that he had been paralyzed in the right side six years previously, and made other assertions. He therefore probably was not motor aphasic, though we could not assert that his speech was normal. An old lesion, a cyst, was found confined to each external capsule and claustrum, and yet persistent or complete aphasia seems to have been wanting. The right hemiplegia might be attributed to this cyst in the left external capsule, but inasmuch as a small area of degeneration was found in the middle of the foot of the left cerebral peduncle, it seems possible that some other lesion in the left cerebral hemisphere has been overlooked. The lenticula on each side, to the naked eye, seems to have escaped.

This case, after all, is of chief importance as illustrating the points made when discussing the views of Edinger and others as to the embryonal, and even later, separations of the insula and its subcortex from the basal ganglia, the inference being that they are functionally, anatomically and morphologically distinct.

Case 7. *Right Hemiplegia with Aphasia—Deep Reflexes but* *little changed—Some Hypesthesia—Stereognosis not studied —Aphasia of Motor Type—No Word Deafness—Thrombus of Left Middle Cerebral Artery just above the place where the Vessels to the Basal Ganglia are given off—Softening invoking the Cortex and Subcortex of the Insula, the Foot of the Precentral and Postcentral Convolutions, the Entire Parietal Lobe, the Fibers to the Lower Part of the Callosum, the Optic Radiations to a small extent—The Lenticula and the Temporal Lobe were not implicated in the Lesion.*

A. H., sixty years old, colored, was admitted to the Philadelphia General Hospital December 15, 1904, and died December 29, 1904. The only history that was obtainable was that she had not spoken for five days and since that time had been paralyzed in the right half of the body.

On examination the patient was found lying in bed with her eyes closed, making no attempt to move or talk. The face was drawn to the left over one-half of its extent. There was marked over-action of the occipito-frontalis of the left, while on the right movement of this muscle was very slight.

The eyes were directed anteriorly without deviation; the right pupil was contracted, and apparently immobile. She could not be made to accommodate; the left pupil was fixed; the media of both eyes were very clouded, especially on the left, which seemed to show a cataract. Arcus senilis was marked. She could not be made to move her eyes in any direction when told to do so, although she evidently understood the command, as she made an effort to obey by a slight turning of the head.

The tongue could not be protruded. She opened her mouth when asked to put out her tongue, which was pushed to the left and was slightly tremulous and somewhat coated.

The right arm was apparently completely paralyzed and fell helplessly when raised. The response was very feeble on tapping the triceps and biceps tendons. The right leg, which was paralyzed, gave a slight knee-jerk but no ankle clonus, although there was a suggestion of dorsal flexion of the great toe on plantar stimulation.

She slightly moved her paralyzed foot and leg occasionally when stuck by a pin. Attempts to elicit ankle clonus occasionally resulted in a flexion of the thigh on the pelvis. The left leg gave a very slight knee-jerk. No ankle clonus and no Babinski response were determined. This limb was moved somewhat more when stuck by a pin than was its fellow. There was no positive result on either side to stimulation by the Oppenheim method.

Under date of December 18, 1904, the clinical notes stated that there was no change in the patient's condition. She understood what was said to her, but could not answer. Her bladder and bowels were incontinent. Her pulse was very irregular and she slept most of the time. She was mentally dull. Temperature at this date was still down, but respiration was up to 50; pulse was at 150. The right chest gave bronchial breathing and great impairment about its middle in the midscapular line. Both sides were full of large bubbling and smaller sized rales. She could be aroused by vigorous questioning, but could not answer, although she tried. She was very weak. She died December 29, 1904.

At the necropsy the following pathological conditions were found in the various organs:

Congestion of the lungs; chronic interstitial myocarditis; chronic congestion and interstitial splenitis; chronic congestion and interstitial nephritis with infarction and hemorrhagic and healed infarct of the kidney; chronic congestion and fatty fibrosis of the liver and general arteriosclerosis. The brain and spinal cord were removed for hardening and further investigation.

Later this investigation showed a thrombus occluding the left middle cerebral artery just above where the arteries to the basal ganglia are given off, so that the latter arterial supply was intact. A large area of softening was found which implicated the cortex and white matter of the island of Reil, leaving a narrow margin of white matter intact on the lateral aspect of the lenticula; the latter ganglion was not affected. The foot of the precentral and postcentral convolutions were softened, but the extreme anterior part of the island of Reil escaped. The softening extended very close to and probably slightly involved the optic radiations.

At a higher level and just above the lenticula the softening extended inwards so as to involve slightly the fibers going to form the lower part of the callosum.

The precentral convolution was softened only in its lower part, but the postcentral was more involved together with almost the entire parietal lobe. The temporal lobe was intact.

This case therefore showed very clearly that the arterial supply of the island of Reil is distinct from that of the lenticular nucleus, and that the lenticule may escape although aphasia is pronounced.

Cast 8. *Apoplectic Attack causing Right Hemiplegia and Loss of Speech—Tongue protruded to the right—Right Hemianesthesia—Memory poor—Partial Recovery of Speech—Aphasia Motor in Type—Reflexes exaggerated on the paralyzed side—Linear Hemorrhagic Scar in the Left External Capsule—Lesion Involving also the Insula and Anterior Limb of Internal Capsule, the Lenticula and Posterior Limb of the Internal Capsule.*

E. J., fifty years old, colored, born in Virginia, an expressman, was admitted to the nervous ward of the Philadelphia General Hospital in the service of Dr. Charles S. Potts, on June 12, 1903.

No family history or previous history of importance was obtainable. The patient denied having had syphilis.

Several weeks before admission he fell unconscious in his wagon and remained unconscious for three days. It was then found that the entire right half of his body was paralyzed. At first he was unable to make his wants known by talking, but speech gradually improved. His wife stated that for several weeks before the paralytic attack he had complained of headache.

On June 14th, two days after admission, it was noted that he could not move his right arm and leg, that his speech was slow and his memory apparently poor. In general appearance the patient was lean and weak. The pupils were equal; the light reaction was normal, the accommodation reaction poor. The pulse was intermittent, the chest normal, the abdomen tympanitic.

Under date of June 17th notes were made that he was anesthetic on the entire right side, the anesthesia extending about one inch beyond the median line on the left side. The conjunctiva on the right side was anesthetic. The patient was so stupid that it was difficult to get from him satisfactory answers. He could not see a quarter of a dollar when it was brought toward him from the right, but he recognized it at once when brought from the left, or when it was held in front of either eye. Dr. Howard F. Hansell, one of the ophthalmologists of the hospital, however, reported no hemianopsia. On the right side he could recognize cold, but invariably called *hot* "cold." He could not distinguish any difference between two test tubes containing hot and cold water when they were placed on the right side, but could at once when they were placed on the left side. He complained of much pain when the right arm or leg was moved, but these limbs could be pressed or touched with impunity and he did not complain of pain in these parts while they were at rest.

He had use of all parts of the left arm, although power in it was somewhat impaired. He could also move the left leg properly, but with some diminution of power.

Further examination showed absence of sensation in the right side to above the costal margin, except on stroking with a sharp point. Sensation was also absent in the right arm and upper part of the body, except on very deep sharp pressure. Sensation was present on the left side. The knee-jerk was increased on the right side and still more increased and spastic on the left. The biceps jerk was increased on each side. The tongue, which was tremulous, was protruded slowly and to the right.

On June 22d examination showed that hemianopsia was absent. The Babinski reflex was present on the right; the plantar reflex was present on the left. No ankle clonus was present on either side. The tongue protruded decidedly to the right, with tremor. The naso-labial fold was lessened on the right; the mouth was very slightly drawn to the left. The pupils were equal, contracted to light and very slightly to accommodation.

Arcus senilis was present. The arteries were thickened and tortuous, the right radial being somewhat beaded. The pulse was intermittent, its tension moderate. The heart was irregular in action, the second sound being accentuated, the first sound clear but not proportionately loud. Considerable atrophy and contracture were present in the right hand; the patient complained of pain in moving his right leg. He had frequent micturition and had control of bladder and rectum.

He apparently understood all that was said to him, but was not always able to find words to answer. When the proper answer was repeated to him, he recognized it. There was therefore apparently some motor aphasia.

On July 15th he called the hot test tube "cold" on both sides, and still had complete absence of power in the right arm and leg.

On July 25th it was noted that he had made some improvement during the preceding five days, but on this afternoon he became very suddenly prostrate, dyspnea became marked, and the pulse weak and rapid. He died during this day at a time not mentioned in the clinical record.

Necropsy showed the dura and pia normal. The upper aspect of the right cerebellar lobe was edematous, grayish yellow in color with distinct loss of substance. Inspection of the ventricles was negative. The vessels at the base of the brain were sclerotic. The specimen was removed for hardening and further examination.

Other pathological conditions present were: Fatty degeneration of the liver and myocardium, thrombosis of the pulmonary arteries, chronic pleuritis, edema and congestion of the lungs, chronic nephritis, slight prostatic enlargement, fatty degeneration of the kidneys, especially of the right, deformity of the right foot, and ulcer on the right ankle.

A horizontal section made through the cerebral hemisphere at the level of the upper part of the basal ganglia showed a linear hemorrhagic scar in the left hemisphere in the external capsule. It extended forward as far as the anterior limit of the island of Reil but not beyond, its forks sending off a branch cutting the anterior limb of the internal capsule. It implicated the greater part of the left lenticula, and extended very near to the posterior limb of the internal capsule, although apparently it did not implicate this limb at this level. The cortex of the island of Reil was not involved. The lesion extended downward in the same relative position about 1 cm. (2-5 in.) from this level. The upper part of the scar, in a section from a higher level, involved the posterior limb of the internal capsule, and as the island of

Reil is very short at this level the scar extended forward here beyond the anterior end of the island.

It will be noted that the lesions in this case involved both the external capsule, the anterior limb of the interior capsule, the lenticula, and to some extent the posterior limb of the internal capsule; also slightly the island of Reil. It is therefore difficult to draw any positive inferences with regard to the lenticula; the motor aphasia may have been due to lesion of the external capsule and the island, or to the lenticular lesion.

This man, several weeks after the beginning of his right hemiplegia, had slow speech; he apparently understood all that was said to him, but was not always able to find words to answer. When the proper answer was repeated to him, he recognized it. There was apparently some motor aphasia, but it was by no means complete, and yet a large part of the putamen was destroyed.

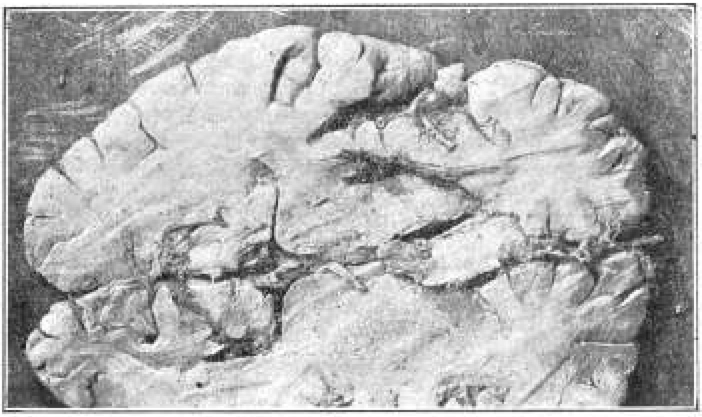

Fig. 8. A hemorrhagic cyst is present in the left lenticular nucleus. Some motor aphasia persisted. (Case 8.)

The occurrence of the hemorrhage into the external capsule of one side is interesting in connection with what has been said of lesions thus located and of the anatomical and functional separation of the basal ganglia from the insula.

Case 9. *Left Facial Paresis—Deviation of Tongue to the Right—Spasticity of Right Upper Limb and of the Lower Limbs—Babinski Response on Both Sides—Spasticity Present and Deep Reflxes Exaggerated on the Left—Incontinence of Urine and Feces—Involuntary Laughing and Weeping—Mentality Dull—Speech Indistinct but no true Motor Aphasia Present—A Small Cyst in the Left Putamen extending into the White Matter of the Insula.*

R. S., sixty-two years old, white, German, was admitted to the nervous wards of the Philadelphia General Hospital October 24, 1905.

Examination showed the left naso-labial fold to be less marked than the right. The tongue protruded to the right. The brow was wrinkled equally well on the two sides. The pupils were unequal, the left being larger than the right. The ocular movements seemed to be preserved, but the patient's mental condition rendered the examination of these movements a somewhat uncertain matter. On attempting to close the eyes, the left was not so well closed as the right, the edges of the left being separated by quite a little distance as compared with the right. The pupils reacted to light and in accommodation.

The patient seemed to have considerable strength in both upper extremities, all movements being preserved. A suggestion of spasticity was present on the right side. The reflexes of the right upper limb were increased; the left were not increased. The patient had lost control over the bladder and bowels. Movements of the legs were restricted and appeared to be spastic, but it was here again, as with the ocular movements, difficult to say how much was paralysis and how much failure to understand. Both knee-jerks were plus and the Babinski response was obtained on each side. The ankle jerk was also much increased on each side. The biceps jerks were present on both sides.

Under date of December 18th additional notes were made as follows: The right knee-jerk was plus; the left plus, plus; the ankle jerk was plus on the right side and also on the left; Gowers' front tap was present on the left side and absent on the right; ankle clonus was absent on both the right and the left sides.

Stimulating the sole of the foot gave very uncertain results. On the right side at times extension of the toes appeared to be present, and at other times, flexion. On the left side the tendency seemed to be towards the extension or Babinski response.

Power in both upper limbs was much diminished. Movements were much better performed with the right upper extremity than with the left. Fine movements were very poorly and clumsily performed. Movements were very ataxic, especially those of the right side. Marked tremor was present when the fingers of each hand were extended. The limbs on the right side were spastic, and contractures were present. The patient could move the right leg, but not nearly so well as the left. Sensation to touch, pain and temperature seemed to be undisturbed.

On December 27th further notes were made. The patient's condition was not so good as it had been. The nurse stated that the patient had occasional spells of laughing and crying; her mentality was not clear; speech was not distinct, and it was only with difficulty that she could be understood. She had no trouble with chewing or swallowing; she had no dribbling of saliva. The pupils were unequal, both being larger than normal, the left larger than the right. Extraocular movements were normal. It was impossible to tell the condition of the light reflex and accommodation and convergence. Apparently the seventh nerve on each side was not involved. Apparently ptosis of the right upper lid was present. The other conditions were as noted in the preceding observations.

The bedside notes contain a statement without date made by Dr. Spiller that the patient's speech was very indistinct, but that she was not aphasic.

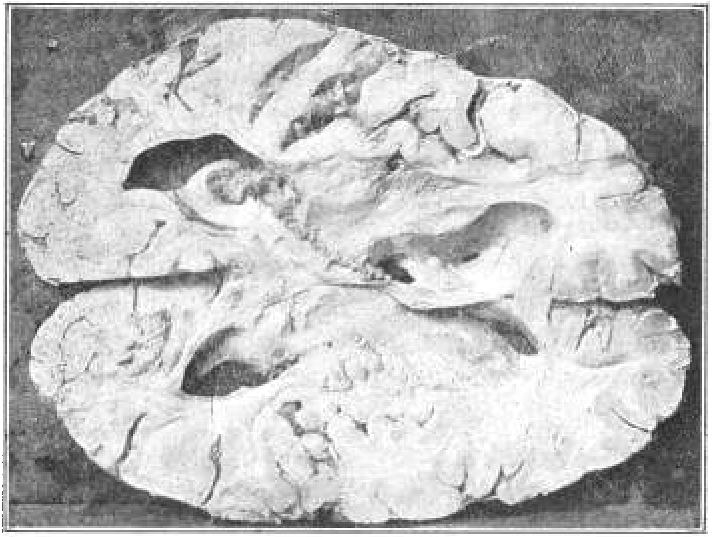

Fig. 9. A small cyst is present in the left external capsule, extending into the lenticular nucleus. The speech was indistinct, but not aphasic. (Case 9.)

On February 4, 1906, while eating her dinner, she suddenly turned to the attendant who was feeding her and said that she had enough. Immediately after this she gasped for breath, became cyanosed and fell over dead.

At the necropsy the pathological findings showed the presence of hypertrophy of the heart; chronic passive congestion of the spleen; chronic interstitial nephritis: passive congestion and fatty infiltration of the liver and general arteriosclerosis. The skull appeared to be thicker than normal. The brain and spinal cord were retained for subsequent investigation.

The gross lesion found was a small cyst 0.5 x 0.5 cm. (1-5x1-5 in.) in the middle of the left putamen in a horizontal section through the upper part of the basal ganglia. This cyst did not extend into the cortex of the island of Reil, but implicated the white matter in the middle of the island.

It would seem in this case that the anarthric or dysarthric affection of speech and the impairment of power and spasticity and exaggerated reflexes on the right side might be fairly attributable to the lenticular and subcortical lesion. The speech disorder in particular is similar to that which seems to have been observed in other cases of limited lesion of the putamen—not a true motor aphasia, but a speech disturbance more nearly allied to that which is seen in pseudo-bulbar paralysis from capsular lesions. The woman was sixty-two, and some of the symptoms may have been caused by senile changes in the brain, but this finding is important, viz., a small cyst interrupting the fibers in the left external and extreme capsules, and extending into the putamen in about its middle portion.

Case 10. *Apoplectiform Attack without Unconsciousness—Speech Lost for Two Hours, then Recovered—Hemiparesis of Right Lower Face—Deviation of Tongue to Right—Paralysis of Right Upper and Lower Extremities—Reflexes increased on right—Sensation not impaired—Destructive Lesion involving the Left Lenticula and both the Anterior and Posterior Limbs of the Internal Capsule—Absence of Persistent Motor Aphasia.*

A. W., white, thirty-six years old, was admitted to the nervous wards of the Philadelphia General Hospital December 27, 1898. The family and previous history of the patient contained no fact of importance, except that her mother died of dropsy at the age of forty-two. The patient used alcohol moderately.

Five weeks before admission the patient had an attack of influenza from which she recovered. Three weeks before admission she was taken with weakness while on the street and felt three times. While unable in these spells to stand or walk, she remained perfectly conscious. Her speech was affected for two hours, during which time she was unable to talk. On attempting to move the right arm or leg she was unable to do so.

Examination showed a fairly well nourished adult. The pupils were equal; the irides reacted to light, and in accommodation and convergence. The extraocular muscles and the eyelids were not paralyzed. The muscles of the right side of the face appeared to be partially paralyzed, this being especially noticeable in the lower part of the face. The tongue deviated to the right on protrusion.

The right arm and leg were completely paralyzed, but there was no limitation of movement in the left extremities. The biceps and triceps jerks were increased on the right side and absent on the left; both knee-jerks were increased. Slight patellar clonus was present on the right side; ankle clonus was absent. The plantar reflexes were increased on the right side. Sensation was nowhere impaired.

The pulse was frequent, small, and fairly strong. The heart sounds were normal; the second sound was accentuated.

Examination of the lungs showed nothing on palpation and percussion, but on auscultation at the right apex was heard a slight prolongation of expiration, especially noticeable at the middle lobe posteriorly.

An occasional note regarding this patient was made between her admission and November 17, 1903, simply showing that the patient was a right hemiplegic. On November 17, 1903, examination showed that the woman was weak and her breathing shallow, but that she was conscious. Both the right upper and lower extremities were completely paralyzed. Knee-jerks were present on both sides, prompt on the right; the Babinski response could be elicited on the right, but was absent on the left; neither ankle clonus nor patellar clonus was present. The tongue protruded slightly to the right. No old contractures were present on either side. Sensation was not impaired.

Notes were made at this date that the patient had not slept well. She was constantly muttering and picking at the bedclothes. She could, however, be aroused and made to answer questions intelligently. Respiration was increased, but not labored. The tongue was heavily coated. Examination of the urine was negative. Resonance was impaired over portions of the middle and lower lobes of the lungs on the right side; breathing was distinctly tubular over an area of the middle lobe. On November 18th the patient developed edema of the lungs; the heart became very rapid and weak. She died November 19, 1903.

The following conditions were found post mortem: Croupous pneumonia with red hepatization and right-sided slight hydrothorax; slight chronic diffuse nephritis and acute parenchymatous nephritis; also chronic passive congestion of liver; slight fatty degeneration of liver; slight chronic thickening of the pia arachnoid; atheroma of thoracic and abdominal aorta.

A horizontal section was made through the left cerebral hemisphere, just at the upper border of the head of the caudatum and thalamus, so as not to remove any of these structures. At this level a cyst was found occupying the entire area of the lenticula and encroaching upon the anterior and posterior limbs of the internal capsule and the white matter of the island of Reil. The cortex of the latter region was intact. The head of the caudatum and the thalamus, so far as could be determined by the naked eye, were not implicated. The cyst did not extend forward or posteriorly beyond the limits of the lenticula. It measured 3.5 cm. in length and 1.5 cm. in width (1 2-5x3-5 in.).

A horizontal section made through the cerebral hemisphere

1 cm. (2-5 in.) below the previous section showed the lower termination of this cyst. It was directly beneath the center of the cyst as shown in the previous section and measured 1.5 cm. long by 1 cm. wide (3-5x2-5 in.). Sections of the spinal cord made by Dr. Weisenburg showed intense degeneration of the right crossed and left direct pyramidal tracts.

The lesion present in this case, although in large part in the lenticula, was not confined to it, extending both into the anterior and posterior limbs of the internal capsule. We cannot therefore draw any positive conclusions as to the part played by the lenticular lesion in the production of the hemiplegia. The case is chiefly of value in throwing some light upon the effect of so large a lenticular lesion on the function of speech. Speech, while affected at first, was soon recovered, and the patient continued to speak intelligently. She was not a motor aphasic.

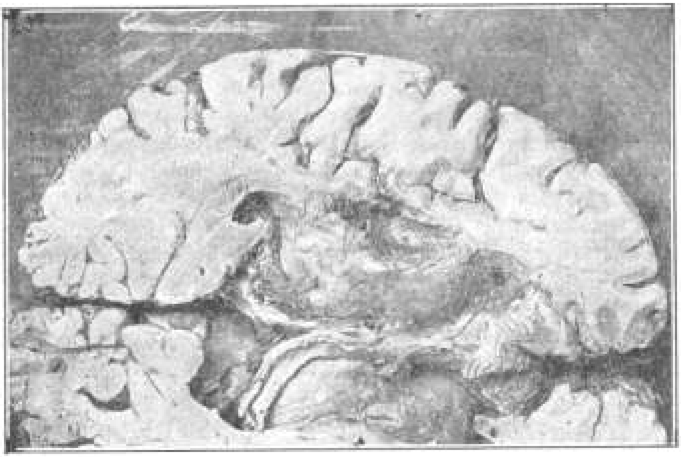

Fig. 10. Complete destruction of the left lenticular nucleus and anterior and posterior limbs of the left internal capsule, without persistent motor aphasia. (Case 10.)

This in connection with the first case of this paper shows that complete destruction of the left lenticula does not necessarily cause complete and persistent motor aphasia. While the notes on the condition of speech in this case are not as complete as might be desired, they indicate very clearly a recovery, in part at least, of the motor speech function, and we have the statement from Dr. T. H. Weisenburg that when a resident in the hospital he frequently conversed with the patient, and that she was able to speak. We cannot conclude, however, that speech was normal.

Case 11. *Aploplectic Attack causing Complete Right Hemi-*

plegia with Hemianesthesia and Complete Aphasia—Disappearance in large part of Speech Affection and Hemiplegia—Persistence of Hemianesthesia—Right Lateral Anopsia—Dyslexia—Partial Word Deafness and Partial Word Blindness—Deviation of Tongue to the Right—Right-sided Spasticity and Contractures and Exaggerated Reflexes—Large Cyst involving Left Extreme Capsule, Claustrum and External Capsule and Destroying the Left Lenticula and Carrefour Sensitif, also involving Optic Radiations—The Anterior Half of the Posterior Limb of the External Capsule and the Anterior Limb of the Internal Capsule not involved.

A case which was first recorded by one of us, Dr. Mills,[28] in a paper which was written in collaboration with Dr. G. E. de Schweinitz, is of some value in connection with the study of the motor and speech functions and the question as to the sensory functions of the lenticula. This patient died in the wards of Dr. F. X. Dercum, in the Philadelphia General Hospital, and the case was made the subject of a paper by Drs. Dercum and Spiller,[29] on permanent hemianesthesia resulting from destruction of the *carrefour sensitif* and the lenticula without implication of the thalamus, the case appearing to demonstrate that the cerebral sensory fibers are located chiefly, if not altogether, in the area of the *carrefour sensitif*, or it may be that some sensory fibers pass through the lenticular nucleus, as held by Edinger. The writers define the *carrefour sensitif*, following Verger, as comprising the last third of the posterior limb of the internal capsule in the most anterior portion of the opto-striate region.

The hemorrhagic apoplectic attack which left the cyst found at necropsy occurred many years before the death of the patient. Immediately after the attack the man was completely paralyzed in the right arm and leg, and also completely aphasic and hemianesthetic. He recovered his speech very largely in about two months and the motor paralysis largely disappeared in about three months. The hemianesthesia remained and was extremely marked up to the time of the patient's death. The man was paralyzed in 1892. Four years later, in 1896, when the clinical report of Dr. Mills and Dr. de Schweinitz was made, he had right lateral quadrant anopsia; absence of Wernicke's symptom; dyslexia; right hemiparesis; partial right hemianesthesia; and partial word deafness and word blindness. His tongue was slightly tremulous on protrusion and deviated a little to the right. With regard to his aphasia, the patient stated at this time (1896) that from the first he knew what he wanted to say, but could not put it into such words as would be understood by his hearers. He could recognize objects, but could not pronounce their names. He could not read writing or print, because everything ran together; he recognized the letters, but could not pronounce them. He understood sentences, but could not read them. In 1896 he had almost completely recovered from his aphasia, although his voice was a little thick and difficult to understand.

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[28] Mills, Chas. K., and de Schweinitz, Geo. E. The Philadelphia Hospital Reports, Vol. III., 1896.

[29] Dercum, F. X., and Spiller, William G. The Amer. Journ. of the Med. Sciences, N. S., 123, March, 1902.

In 1899 there was present a spastic hemiplegia of the right side, moderate in severity, with a moderate degree of contracture of the right arm and spastic rigidity and exaggeration of the knee-jerk on the right side. The right side of the face was only slightly involved. The hemianesthesia appeared to be everywhere complete, and was associated with right-sided homonymous hemianopsia.

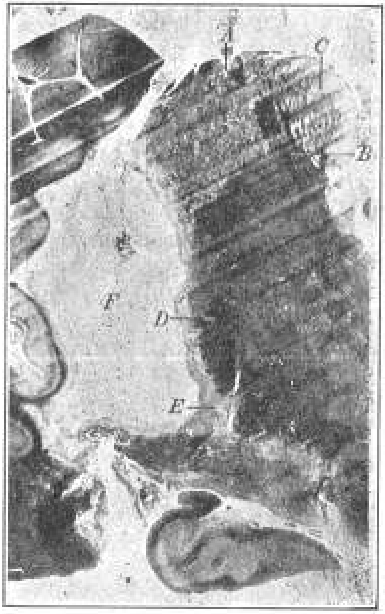

Fig. 11. A. Portion of the lenticular nucleus not destroyed. B. Anterior limb of the internal capsule. C. Head of the caudate nucleus. D. Posterior limb of the internal capsule. E. Carrefour sensitif. F. Cyst in the lenticular nucleus. (Case 11.)

"A cavity, probably resulting from an old hemorrhage, 2.8 cm. in depth from above downward, and 2.5 cm. in its longest diameter, (1 1-8 in. x 1 in.), was found in the extreme capsule, claustrum and external capsule of the left side. The left lenticula.appeared to be almost destroyed and the extreme posterior part of the posterior limb of the internal capsule and the optic radiations were involved in the destructive lesion. The anterior half of the posterior limb of the internal capsule was not implicated in the cyst. The anterior limb of the internal capsule was entirely normal. The thalamus was not implicated at all in the cyst, and the external medullary lamina of the thalamus was distinct and normal in appearance. The external nucleus seemed to be of about normal size. A transverse cut passing through the hypothalamic region showed the anterior half of the posterior limb of the internal capsule apparently normal. The lenticular nucleus, except in its extreme ventral portion, was destroyed.

"The portion of the thalamus that showed atrophic change was the pulvinar, and this alteration was the result of the destruction of the optic radiations."

In addition to the macroscopical examinataion of the speecimen in this case, careful microscopical investigation by serial sections was also made, this confirming the fact that the anterior half of the posterior limb of the external capsule and the entire thalamus were not involved in the lesion, while the *carrefour sensitif* and the lenticula, with the exception of its most ventral portion, were destroyed.

It would seem fair in this case to refer the moderate spastic hemiparesis which persisted until the death of the patient partly to the lenticular lesion, as the motor subdivision of the posterior limb of the internal capsule was only a little involved. The early aphasia which disappeared in two months was probably due to compression or partial destruction of some part of the lenticula concerned in speech phenomena, which must be the anterior portion, the compression of the neighboring speech areas in the temporal lobe and to the destruction of the insula. We are inclined to refer the permanent hemianesthesia chiefly to destruction of the *carrefour sensitif*, although we do not deny that sensory fibers may pass through the lenticula. The disturbance of both phonation and articulation which is referred to in the statement that his voice was thick and difficult to understand, was probably dependent upon the lenticular lesion, as the genu and anterior half of the posterior limb of the internal capsule were not destroyed.

Both from our study of the literature of the subject and from the analysis of the personal cases given in this paper, we find is somewhat difficult to form positive conclusions as to the symptomatology of lesions of the lenticular zone.

We believe, however, that a few general conclusions may be drawn:

1. Lesions restricted to the lenticula apparently do not cause sensory symptoms;

2. Motor symptoms probably result from lesions situated in certain parts of the lenticula: speaking generally, the lenticula may be regarded as a motor organ;

3. Anarthric or dysarthric speech disorders result from lesions of some portion of the left lenticula, which probably contains centers which are concerned with the movements which make speech possible;

4. Destructive lesions of certain portions of the lenticula probably cause a paresis of the limbs or face;

5. The paresis or paralysis caused by destructive lesions of the lenticula differs from that produced by capsular lesions, the impairment of power not being so severe and not being so characteristic in the former as in the latter case;

6. The paresis or paralysis which is caused by lenticular lesions differs from that produced by cortical lesions in that it is less likely to be dissociated; although dissociated lenticular paresis may occur;

7. While the loss of power which results from a destructive lenticular lesion is permanent, it is usually not intense;

8. Persistent true motor aphasia, as this form of speech disorder is generally understood, is not caused by a lesion restricted to the lenticula, no matter what its size or destructiveness;

9. The insula, cortex and subcortex play an important part in speech phenomena, one entirely different from that played by the lenticula and the internal capsule;

10. The insula is a part of the cortical motor center for speech, Broca's convolution probably forming with the insula the entire cortical motor center for speech;

11. Motor aphasia may be present without a lesion of the left third frontal convolution;

12. The lenticula forms too large a portion of the cerebral hemisphere to be regarded merely as a vestigial organ.